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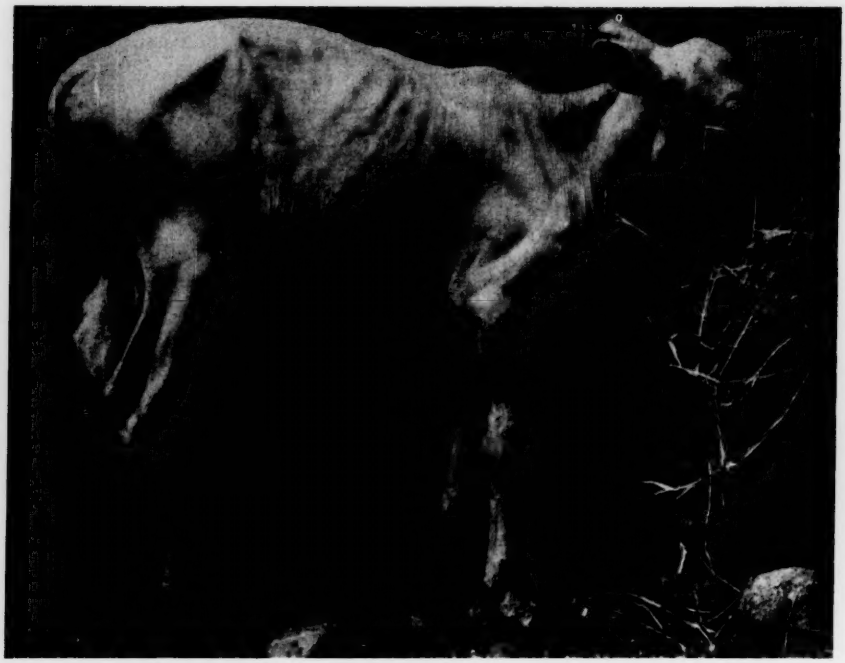

GENETIC HAIRLESSNESS IN GUERNSEY CATTLE

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Satin Rabbits

"Jittery" Chickens

Tailless Cocker Spaniels

Sex-Determination in Spinach

VOLUME 44 MAY-JUNE, 1953 NUMBER 3

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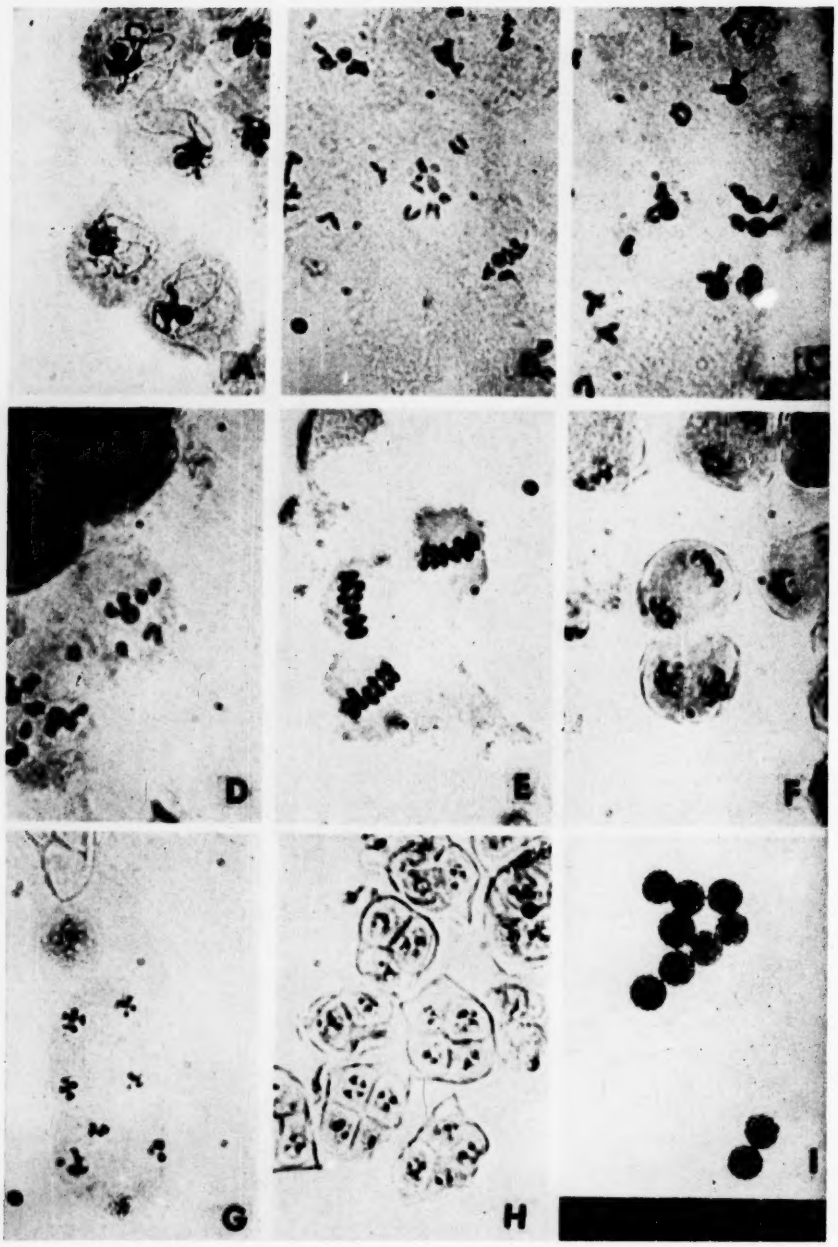


MEIOSIS IN *SPINACIA OLERACEA* L.

Figure 1

A—Pachytene; *B*—Diakinesis; note that two pairs of chromosomes are associated with the nucleolus; *E*—First Metaphase; *F*—Interkinesis; *G*—Second Anaphase; *H*—Tetrads; *I*—Pollen. Each division of the scale represents 10μ.

A NEW HYPOTHESIS EXPLAINING THE GENETICS OF SEX DETERMINATION

In *Spinacia oleracea* L.

W. P. Bemis and G. B. Wilson*

THERE is good reason for believing that the genetic mechanism controlling sex expression does not differ from that controlling other heritable characters.[5] Therefore, the difference between two organisms exhibiting contrasting sexual potentials must be considered essentially genic. The major difficulties in studying sex expression usually arise from the problem of discrete phenotypic classification, especially where intersexes are involved.

According to the "Balance Theory" of sex determination[3, 4] two organisms which differ with respect to maleness and femaleness possess different "dosages" of male and female determinants. Where this difference is consistent, one or the other of the two sexes may be presumed to have some mechanism leading to heterogametic production. In many cases the difference between sexes is cytologically recognizable but in others, especially where certain dioecious plant species are concerned, no obvious difference can be noted.[1]

The present paper is concerned with preliminary investigations into the nature of sex determination in the normally dioecious species, *Spinacia oleracea* L. Most commercial varieties of spinach segregate quite clearly into obvious male and female plants in a 1:1 ratio, leading to the conclusion that a precise genetic mechanism is operating. However, all varieties produce a small percentage of plants exhibiting various degrees of intersex expression, (Figure 2) which in turn raises several questions which may be expressed conveniently as follows:

1. Do intersexes represent fluctuations induced by environment in one or both of the primary sex types?
2. Is there an obvious chromosomal difference between male and female fluctuations in which may occasionally lead to the formation of intersexes?
3. Do the intersexes represent comparatively rare recombination types?

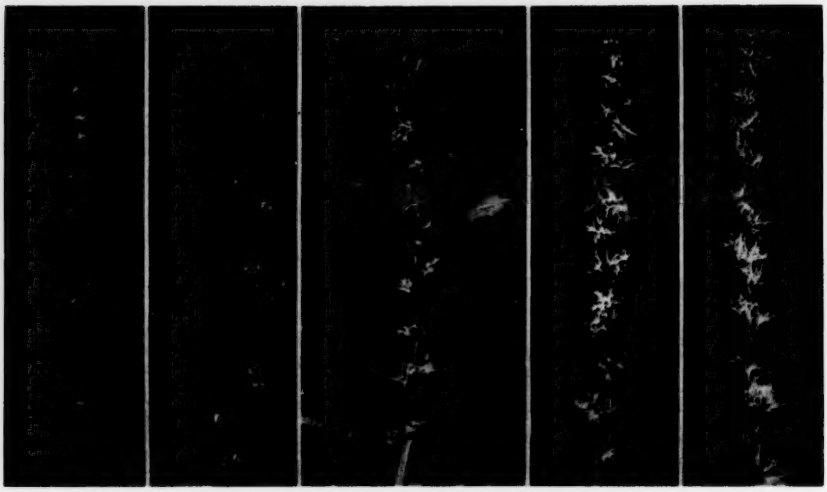
1. Environmental Influence on Sex Expression

A number of preliminary experiments were conducted with several varieties in order to determine whether such factors as time of planting and spacing greatly influence sex expression either in the direction of altering ratios or changing the frequency of intersex production.[9] No significant effect of any kind was noted. Furthermore, the fact that the Canadian Department of Agriculture has produced a true breeding intersex form by selection may be construed as strong evidence that sex expression is primarily under genetic control. Seeds of this variety were supplied by Mr. J. J. Jasmine of the Canadian Department of Agriculture and were grown along with our own selections for comparison. The several plantings of both types gave rise to remarkably uniform intersex types. However, comparisons between field and greenhouse plantings indicated that environment plays a significant role in the rate of development of flowers in different intersex types especially those which do not differ greatly from pure males and females. Nevertheless, intersex types were usually identifiable as such under either condition.

2. Cytology

Extensive cytological studies on *S. oleracea* have been made by several investigators and

*First Assistant in Vegetable Crops, Department of Horticulture, University of Illinois and Associate Professor of Botany, Michigan State College, respectively. This paper is based primarily on a thesis presented by the senior author to the School of Graduate Studies of Michigan State College in partial fulfillment of the requirements for the Ph.D. degree. Contribution No. 53-15 of the Department of Botany and Plant Pathology, Michigan State College, East Lansing, Michigan. The authors wish to express their appreciation to Mr. P. G. Coleman, Experiment Station Photographer, Michigan State College, who took the original photographs and prepared the plates.



SEX TYPES IN *SPINACIA OLERACEA* L.

Figure 2

A—"Pure" male; *B*—Male intersex; *C*—Intersex with androecious and gynoecious flowers about equal; *D*—Female intersex with terminal flowers androecious; *E*—"Pure" female.

all seem to agree that there is no obvious heteromorphic pair of chromosomes associated with either primary sex type.[6, 7, 10, 12] Araratjan[2] reported a heteromorphic pair for *S. tetandra*, but was unable to find a similar pair in *S. oleracea*. As a part of the present work, somatic complements of all major sex types were examined and detailed meiotic studies were made of two unrelated intersex lines as well as "pure" males of a standard commercial variety. The karyotype, based on both somatic and meiotic figures, appears in Figure 3. A series of photomicrographs illustrating meiotic stages appears in Figure 1. In no case was any evidence found of chromosome differences, either with respect to behavior or type between different sex forms. With the exception of minor differences in relative chromosome sizes, the results are consistent with those of other investigators. Although there is no evidence of the presence of a heteromorphic pair of chromosomes, the genetic data discussed below indicate the presence of a differential pair in one of the sexes.

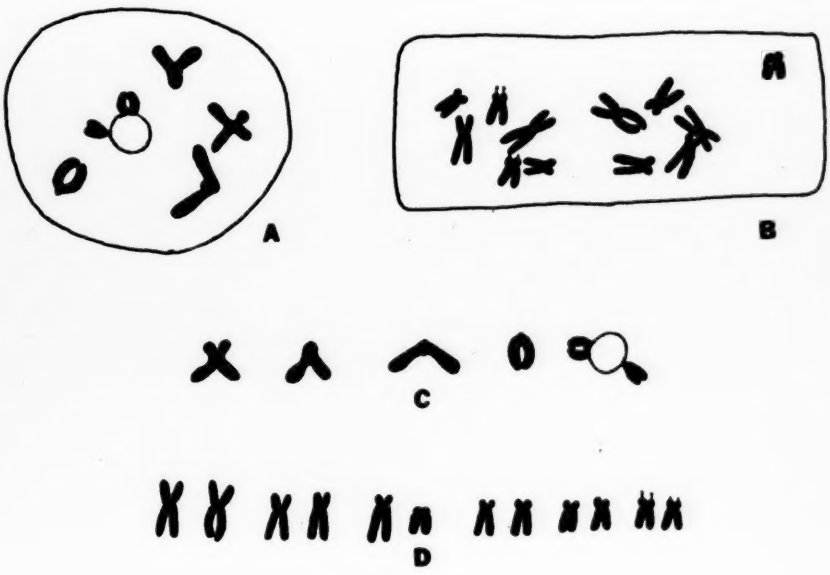
3. Genetics

The primary material from which the genetic data were ultimately obtained consisted of seed collected from open pollinated intersex plants of various types in a planting of Long Standing Bloomsdale. From the resultant progeny, intersex plants of various kinds were selected and selfed for three successive generations. The fourth generation was carefully scored and the data analyzed in terms of several hypotheses which attempt to explain the genetic mechanism of intersex formation.

It is apparent that any satisfactory hypothesis must be capable of explaining the following observations:

1. General maintenance of a 1:1 sex ratio
2. Occurrence of a variety of intersex strains
3. Maintenance of pure breeding intersex strains
4. Segregation of the progeny of different intersexes into
   (a) males, females, and intersexes;
   (b) females and intersexes only and
   (c) males and intersexes only.

In conformity with the above conditions it appears that, in addition to the standard X/Y mechanism, there may be a pair of closely linked autosomal loci, one of which carries the genes A or a for maleness and the other the genes G or g for femaleness. Accordingly, as long as the two potentials are balanced, segregation will be into males and females without intersexes. However, occasional crossing over in the heterozygote, AG/ag, will result in a variety of intersexes some of which segregate and others breed true. For example, according to expectation a genotype such as X/X, Ag/Ag would be a true breeding intersex while a genotype such as X/Y, aG/ag on selfing would segregate into females, intersexes, male intersexes and males. A theoretical ratio of five males to two females to three intersexes may be obtained by combining the males and the male intersexes. Such a ratio was actually obtained from the selfed progeny



KARYOTYPE OF *SPINACIA OLERACEA* L.

Figure 3

A and *C*—Chromosome pairs at diakinesis; *B* and *D*—Somatic complements.

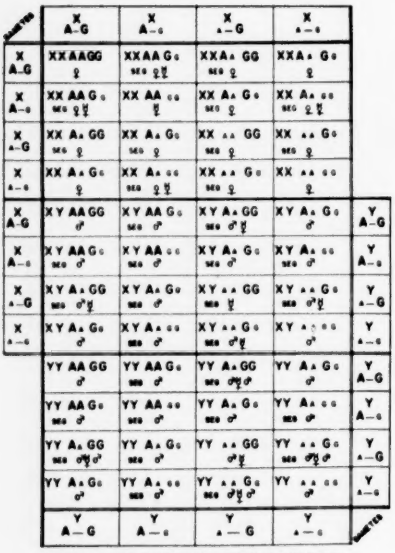
of a plant identifiable as an intersex with male flowers predominating (Table 1).

The proposed hypothesis assumes that *Y/Y* types of individuals are both viable and normal in growth, an assumption which fits the data so far obtained. In the absence of a detectable heteromorphic pair of chromosomes there is no *a priori* reason why this should not be true since the difference between the *X* and *Y* chromosomes may very well be confined to a short segment containing a crossover suppressor. Figure 4 shows all the possible genotypes and Table I gives some observed segregations for several types of selfed intersexes and crosses.

The hypothesis outlined here differs only in minor details from that proposed by Sugimoto[11] who suggested the presence of an autosomal factor influencing sex in addition to an *X/Y* mechanism. According to his hypothesis, *ZZ* is female determining, *zz* male determining and *Zz* intermediate. The only advantage of the hypothesis proposed here lies in its ability to explain the presence of a number of observed intersex types on a genetic rather than on an environmental basis. Despite the fact that the studies on the behavior of all noted types are incomplete, more genotypes are present than can be explained readily on the basis of a single factor hypothesis.

Discussion

While there are no visible chromosomal differences between the various sex types of spinach, the consistent 1:1 male to female ratio, as well as the genetic behavior of the several intersex forms suggests the presence of a heterogenic chromosome pair concerned with the primary sex differential and thus identifiable as an *X/Y* system similar to that in many animals and some plants. By assuming crossing over within the differential segment it would also be possible to explain almost any type of intersex. However, such a system appears to be incapable of explaining some of the observed segregations. The data presented as well as those of previous workers demands the presence of autosomal factors of considerable potency. Both the types of intersexes and their genetic behavior indicate that there is a minimum of two such accessory factors with two allelic forms. The assignment of suitable potentials to these factors together with the assumption of relatively rare crossing over between them may account for both the characteristic stability of the species with regard to sex segregation as well as for the occasional appearance of a wide range of both segregating and stable intersex types.



THE POSSIBLE GENOTYPIC COMBINATIONS

Figure 4

The chart shows all possible genotypic combinations based on the hypothesis presented in the text. Expected sex type and genetic behavior is indicated.

Sex determination in spinach appears to fit the general "Balance Theory" of Bridges[3, 4] and others with a mechanism superficially similar to many others, but more or less unique in details. From the evolutionary viewpoint it would seem quite possible that the unisexual habit of the species could have arisen from bisexual ancestors and under selection pressure, either natural or human, can revert quite as easily.

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TABLE I. Observed and Expected Segregations from Various Types of Intersexes

Pedigree Number	Assumed Genotype		Sex Types — Female	Intersex	Male	χ^2	p
		obs.		49			
XF-14-11-15-1	XX AA gg	exp.		49			
		obs.		58			
XF-14-11-15-4	XX AA gg	exp.		58			
		obs.		112			
XF-14-11-15-6	XX AA gg	exp.		112			
		obs.		65			
XF-14-11-7-21	YY aa GG	exp.		65			
		obs.	1	90			
XF-14-11-7-24	YY aa GG	exp.		91			
		obs.	17	55	24	0.074	0.96
(XF-3-2) × (XF-3-2)-46	XY aa Gg	exp.	18	54	24		
		obs.	10	59	21	4.182	0.13
(XF-3-2) × (XF-3-2)-45	XY aa Gg	exp.	17	51	22		
		obs.	9	48	25	3.249	0.20
(XF-3-2) × (XF-3-2)-48	XY aa Gg	exp.	15	46	21		
		obs.		78	32	0.981	0.32
XF-14-11-7-22	XY aa GG	exp.		82.5	27.5		
		obs.		42	19	1.415	0.21
(XF-3-2) × (XF-3-2)-47	XY aa GG	exp.		46	15		
XF-14-11-15-11	XX AA gg	obs.	39	39		0.000	0.99
FPI #164965	XY Az Gg	exp.	39	39			
XF-14-12-8-34	XX aa gg	obs.	53		41	1.532	0.21
FPI #164965	XY Aa Gg	exp.	47		47		
XF-14-12-8-39	XX aa GG	obs.	1	43	41	0.105	0.72
(XF-3-2) × (XF-3-2)-40	XY aa gg	exp.		42.5	42.5		
XF-14-11-7-19	XX aa gg	obs.		93	4		
XF-14-11-15-6	XX AA gg	exp.		97			
XF-14-12-8-35	XX Aa gg	obs.	31	6	26	12.500	
FPI #164965	XY Aa Gg	exp.	31	16	16		

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(*Received February 18, 1953*)

◆

SEGREGATION OF THE SATIN GENE IN THE RABBIT

Roy Robinson*

Castle and Law[1] have reported upon the recessive monogenic inheritance of the coat type known as satin, so called because the coat possesses a characteristic glossy appearance. The mutation may appropriately be termed satin due to the most obvious feature induced in animals homozygous for the gene. These authors state that linkage tests with numerous other known mutations of the rabbit gave completely negative results. Recombination frequencies in the F_2 indicated free assortment between satin (*sa*) and the following genes: albinism (*c*), angora hair (*L*), non-extension of black pigment (*e*), furless (*f*), Gillet rex coat (*r*-1) and Normandy rex (*r*-3). Testerosses failed to reveal traces of linkage with: agouti (*A*), English spotting (*En*) or H_1 blood agglutinogen. Satin also appears to be inherited independently of brown pigment (*b*).

Apparently the satin mutation is inherited independently of the above ten mutations which are marker genes for eight segments of a chromosome. However, as Castle and Law point out, before the satin gene can be labeled as another independent chromosome segment, many additional mutations remain to be tested for linkage against the gene, (see, e.g., Castle and Sawin,[2] and Sawin, Griffin and Stuart[3]). In particular, due to their established genetic relationships of complete or incomplete independence, the genes dilution (*d*), wide-band (*w*), Viennese white (*v*), German short hair (*r*-2), brachdactylia (*br*), antigen A (*an*) and atropinesterase (*at*), require investigation. This note records data upon the segregation of *sa* with *d* and also with sex.

Although the satin mutation has been known in America since the early 1930's, importation to Britain of animals carrying the gene did not occur until about 1947, when several breeders introduced the stock. These animals were usually albinos and the males were freely mated with many British breeds, including varieties homozygous for dilution. The F_1 were either wild grey or black in color. The F_2 generation of the latter crosses would yield data upon the segregation of genes *sa* and *d*; accordingly the writer appealed for breeding results. Six breeders responded, supplying details of eight litters. In addition matings were performed by the writer.

Table I displays the two respective groups of data (top row from breeders; lower row personally collected) and the total. To enable a test on homogeneity to be applied, the two sexes were pooled

*St. Stephens Road Nurseries, West Ealing, W. 13, England.

for each of the other classes and satin albinos were merged with the ordinary albinos. This leaves five classes and a homogeneity χ^2 calculated for the data failed to reveal significant heterogeneity ($\chi^2_4 = 4.21$, $P = .3$-$.5$), so we can proceed with analyzing the total segregation.

It is regrettable that approximately one-quarter of the total number of individuals could not be used for the prime purpose of testing for independent segregation of the genes *sa* and *d*, due to the epistatic nature of albinism. However, some 90 colored animals placed into eight classes could be utilized and the resulting analysis is as follows:

Item	χ^2	n	P
Sa	3.33	1	.05 - .1
d	.13	1	.7 - .8
♂	1.11	1	.2 - .3
Sa - *d*	.83	1	.3 - .5
Sa - ♂	.13	1	.7 - .8
d - ♂	3.33	1	.05 - .1
Sa - *d* - ♂	.65	1	.3 - .5
Total	9.51	7	.2 - .3

The total number of animals, however, can be utilized to detect departures from

TABLE I. F_2 generation of *Sa* and *d*

Colored								Albino			
Normal				Satin				Normal		Satin	
Black		Blue		Black		Blue					
♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
18	10	4	5	2	0	0	3	6	6	1	2
17	14	3	4	5	3	1	1	4	7	0	2
35	24	7	9	7	3	1	4	10	13	1	4

random segregation of the satin gene with sex and albinism, yielding the following analysis:

Item	χ^2	n	P
C	.10	1	.7 - .8
Sa	4.08	1	.05 - .02
♂	.14	1	.7 - .8
C - *Sa*	.03	1	.8 - .9
C - ♂	2.21	1	.1 - .2
Sa - ♂	.41	1	.5 - .7
C - *Sa* - ♂	.14	1	.7 - .8
Total	7.11	7	.3 - .5

The two foregoing analyses failed to suggest any significant association between *sa* with *d* or with sex. Nor for that matter did the sample disclose signs of association for any of the factors considered, either individually or coupled with others. We must conclude, therefore, that the satin gene is inherited independently of all other known mutations with which it has so far been tested. The analysis, however, reveals a probable significant deficiency of satin individuals which may indicate a slightly lower viability of the type, a point noted by Castle and Law for their stock.

I wish to thank Mrs. E. Coles, late secretary of the British Satin Rabbit Club for valuable assistance in collecting information for this study during her term of office.

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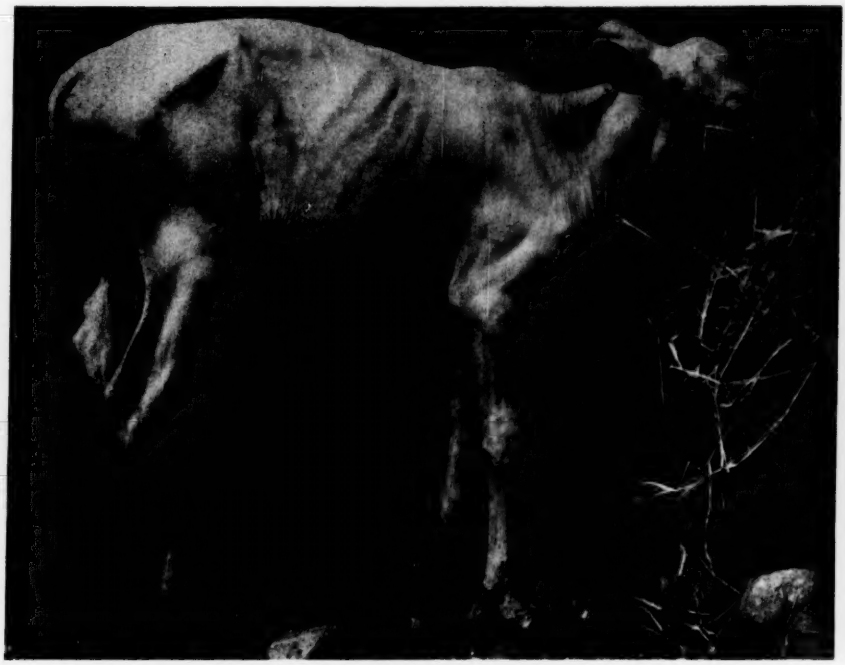
VIABLE GENETIC HYPOTRICHOSIS IN GUERNSEY CATTLE

F. B. Hutt and L. Z. Saunders*



"HAIRLESS" CALF AT EIGHT WEEKS

Figure 5

The tip of the tail carries a normal switch, but elsewhere only scanty hair is present. Little tufts of it are evident at the umbilicus and on the dewlap.

ALTHOUGH varying kinds and degrees of "hairlessness" in cattle have been reported, the recent occurrence in a nearby Guernsey herd of two calves remarkably deficient in hair provided an opportunity to get some facts about a type of defect concerning which little information has been previously available. These are given herewith.

Description

A. Calf. One of the calves, a female, was examined at eight weeks of age. At first glance it appeared to be completely hairless (Figure 5), but on closer examination hair was found on several parts of the body. The last six or seven inches of the tail carried an apparently normal amount of hair, although the rest of the tail was bare. The ears had some hair on the inner surface, but none on the outside or on the edges. There was hair on the eyelids, both upper and lower, on the lips, also a few long hairs on the dewlap and around the umbilicus.

Very fine, short hairs were sparsely

*New York State College of Agriculture and New York State Veterinary College, respectively, at Cornell University, Ithaca, N. Y. The authors are indebted to Dr. R. E. Habel of the New York State Veterinary College, who kindly translated the pertinent part of Liutikov's paper from the Russian.

"HAIRLESS" HEIFER AT 2¾ YEARS

Figure 6

This is the same animal as in Figure 5, but her white spotting is much more distinct. The tail is well-haired at its tip, and a tuft of hair is evident on the dewlap. Hooves and horns are well developed.

scattered over the sides and back of the calf. All of these were irregularly curled and misshapen, none exceeding 2 cm. in length. These fine hairs had been acquired after the calf was a month old. Hair had apparently been developed during gestation, for the owners, who had seen the calf born, said that at the time of birth the hair was peeled back, almost like a fleece. Later "two handsful" of it were expelled. At birth the calf had had a coat of hair on the belly, but that was lost within a month.

The calf's teeth, hooves and horn buds were normal. This fact is important because in two other types of hereditary hypotrichosis in cattle, the teeth are defective or absent.

According to the owners, the deficiency of hair at birth in the other calf, a male, was not so extreme as in the heifer calf. At first it had hair except on the legs and neck, but this coat was lost within a few weeks of birth. It was kept under observation in the clinic of the Veterinary College for several days, during which time it appeared normally bright and showed a healthy appetite. Palpation of the thyroid failed to reveal any sign of enlargement. The teeth and hooves were normal. Some hair was present on the end of the tail and in the ears; elsewhere it was either absent or extremely sparse and fine. The calf was destroyed for autopsy at 10 weeks of age. No lesions were found other than the hairless skin. The thyroid gland was studied histologically and it appeared to be normal for a calf of that age.

B. Adult. The calf described first was not seen again until it was 2 years, 8 months and 13 days old. At a distance it then looked like any other Guernsey heifer, with white-spotting clearly evident in the skin, and the horns normally developed (Figure 6). The tail had enough long hair on the terminal portion to show a normal "switch."

On closer inspection, the heifer was found to have normal hair inside the ears (some of it up to five inches long), some hair between the toes and on the lower portions of the legs, but elsewhere only scraggly hairs, short and curled, sparsely scattered over the body surface. Some areas up to 10 inches in diameter had no

SECTION THROUGH HAIRLESS SKIN

Figure 7

This section, perpendicular to the surface, shows that the hair follicles are shallow and lack the papillae that normally protrude upward from their bases. They contain ony keratinous material, no hair.

hair at all. There were hairs on the eyelids, but fewer of them than in normal cows. As Figure 6 shows, there was a little patch of hair on the dewlap. The short scraggly hairs over the body were unpigmented, even when arising from pigmented skin. The longer and apparently normal hairs inside the ears were mostly red, but some were black.

It is clear from these descriptions that, although cattle showing this condition will inevitably be labelled as "hairless," that term is hardly correct because there is considerable hair on several parts of the body. For that reason it seems more accurate to designate it as hypotrichosis.

Histological Basis

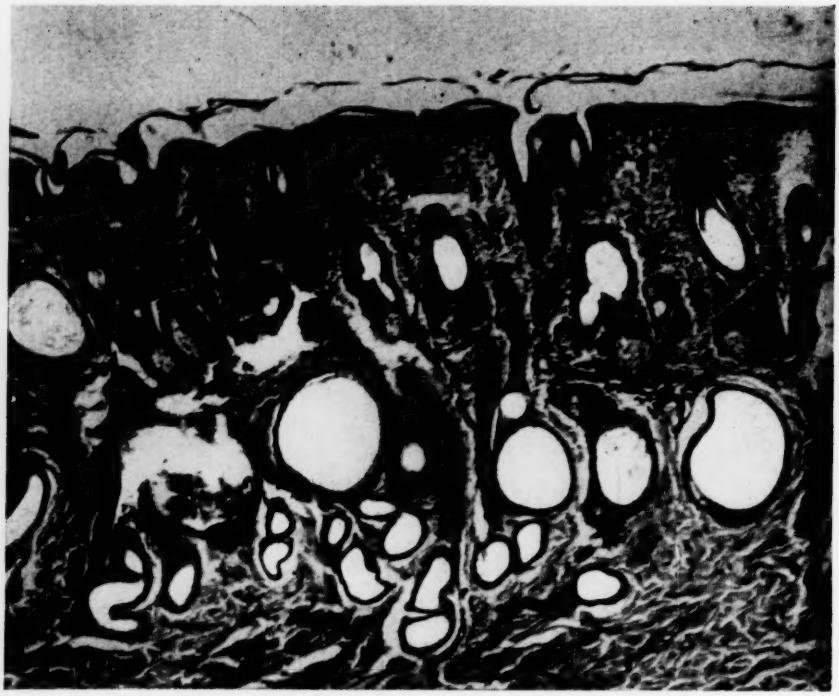
There was no apparent diminution in the number of follicles, but most of them lacked the vascular papilla which normally projects into the bottom of each follicle (Figure 7). Hairs were almost entirely lacking. A few of the follicles contained thin plugs of keratin, apparently derived from cornification of the epithelium lining the lumen. A number of follicles had lost their communication with the surface of the skin during development. Some of these appeared as dilated cysts, often containing keratin (Figure 8), while others appeared as tubes, shaped like boomerangs, with both ends directed toward the subcutis.

A few follicles that appeared normal were scattered among the others, but even these were producing only thin plugs of keratin rather than normal hair fibres. Most of the follicles were completely empty.

The abnormal follicles lacked differentiation of their lining epithelial cells. Cells normally found only in the outer epithelial sheath constituted the only type of epithelium present (Figure 9*B*). This undifferentiated structure was in striking contrast with that of a normal hair follicle (Figure 9*A*), which has three concentric layers of different types of cells (Henle's layer, Huxley's layer and cuticle of the inner sheath) in addition to its outer epithelial sheath. In the abnormal follicles, the absence of differentiation was constant, both in cross sections and in longitudinal sections, and at various levels in the follicles.

Apart from the abnormal follicles, the most striking histological feature of the hairless skin was cystic dilatation of the tubular (sweat) glands (Figure 8). (These structures are commonly called sweat glands, but that name seems hardly correct for a species like the ox in which the glands do not produce sweat.) The cystic condition of these glands suggested that their secretory ducts ended blindly without reaching the surface of the skin, but this possibility was not confirmed by tracing the course of the individual glands in serial sections.

The sebaceous glands were normal in appearance and were present in normal numbers.



HAIRLESS SKIN AT DEEPER LEVELS

Figure 8

This section, which is tangential to the surface, shows the cystic dilatation of the tubular (sweat) glands in the dermis, and some cystic hair follicles above them.

The fact that they were not affected may be due to the fact that many of these glands are "independent" glands in cattle, *i.e.*, they do not empty their secretion into a hair follicle. Hence a developmental anomaly could affect the follicles without involving the sebaceous glands.

Genetic Basis

Since these abnormal calves were both born in a herd of purebred, registered Guernseys, it was easy to trace the relationships of the animals that produced them. These are shown in Figure 10.

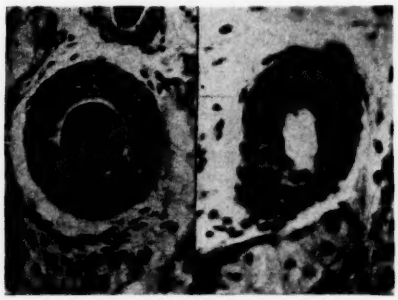
Both calves were sired by the same bull, and the dam of one was the daughter of the cow that produced the other. Since all three parents were normal, and one calf a bull, the other a heifer, the abnormality was evidently caused by an autosomal, recessive gene in the homozygous state. As the pedigree shows, the sire and the cow III-2 were related, and the causative gene is most likely to have been transmitted to both by the sire's dam, I-3. The sire, III-3, had 14 calves by unrelated dams, and all of these were normal.

At the owners' request, names and herd-book numbers of the animals indicated in the pedigree in Figure 10 are not published. These are on file and can be made available if the occurrence of similarly "hairless" calves in Guernsey herds elsewhere should make a study of relationships desirable.

When the first of these two calves appeared, the suggestion was made to the owner that the hairless condition might have resulted from lack of iodine in the dam's diet, but the owners considered this unlikely as they had been feeding iodized salt regularly. Moreover, other calves born about the same time were normal. With the arrival of the second hairless calf, its close relationship to the first left no doubt that the abnormality was caused by inheritance and not by anything in the environment.

Secondary Effects of the Mutation

One type of genetic hypotrichosis in cattle is lethal. It seems desirable, therefore, to em-



NORMAL AND HAIRLESS FOLLICLES
Figure 9

Photomicrographs, at high magnification, of transverse sections showing, *A*—a normal follicle (from a normal calf) with hair in the lumen and concentric layers of different types of cells; *B*—a hairless follicle with no cellular differentiation.

phasize the fact that the mutation occurring in these Guernseys is not. The calf for which we have the best record had the protection of a special blanket during its first few weeks of life and was warmed with an infra-red lamp on specially cold occasions during that period. Since it was born on January 20th, such precautions seemed desirable, and it is possible that without them the calf might not have survived. During its second and third winters no such extra cover was provided.

This calf was born at full term and grew as well as any normal calf. At eight weeks it weighed 95 lbs. Although later weights were not available, it seemed only slightly smaller than normal when seen again at about 2¾ years of age. It ate the same feed that the other cattle ate. The owners said that the heifer had come in heat several times, but had not been bred because they did not want to reproduce the defect in their herd.

Each spring and summer, precautions were taken to expose the animal gradually to increasing amounts of sun. This successfully prevented injury from sunburn in the first two years and the animal ran outdoors much of the summer, except when the flies were bad. In the third spring, the heifer got too much sun at one dose and was severely burned in the areas of white skin on the shoulders. Scars from the blisters thus developed were still evident in October.

Similar Cases

The almost complete lack of hair observed in a Holstein calf by Rosenberger[12] was described with sufficient details and illustrations to indicate that the condition was the same as that in these Guernseys. The animal was sensitive to cold, also to touch, but was thriving at nine months of age. No genetic or histological studies were reported.

The calf reported by Surrarrer,[14] with brief description but a good picture, resembled these others in most respects. Two such calves were sired by the same bull, which was a mixture of three breeds.

It seems probable from the descriptions available that independent occurrences of the same mutation were responsible for these cases and for ours.

Other Kinds of Hypotrichosis in Cattle

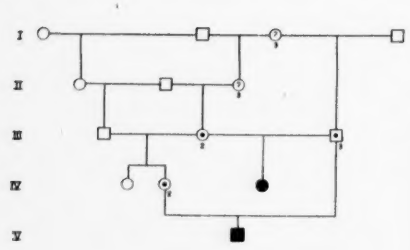
Various kinds and degrees of "hairlessness" in cattle have been reported, but some writers on the subject have failed to differentiate among the several genetically different conditions that fall in this general classification. It seems desirable, therefore, to point out briefly the distinguishing characteristics of each different mutant type. In so doing, we venture to suggest names by which the mutations may be differentiated, all of which follow closely the names used by the original investigators. (We resist the temptation to assign symbols for the causative genes. Experience recently gained in sifting the welter of conflicting usages in the literature to assign symbols, with due respect for priority, to the genes of *Gallus gallus* prompts us to leave to some more devoted follower a similar labor of love for *Bos taurus*.)

1. *Lethal hairless.* This name was used by Mohr and Wriedt,[10] who first studied the mutation. It has also been called *hypotrichosis congenita*. Homozygous calves are born alive at full term, but die within a few minutes after birth. They have hair only on certain sharply demarcated areas, including the muzzle, eyelids, ears, tail and pasterns. Teeth and hooves are normal. In the hairless skin, development of the hair follicles is arrested at an early stage, the sweat glands are cystic and fat cells are lacking in the subcutaneous tissue. It is a simple, autosomal, recessive character.

This mutation was originally found in Holstein-Friesians in Sweden, but it cropped out also in related stock in Germany, where it was reported by Eisele.[4] A hairless calf showing what appears to be the same condition in some other breed was briefly described, with illustrations of both the calf and a section through the skin, by Følger.[5] The lethal hairlessness found by Shibata and Ishihara[13] in Japanese cattle may be the same mutation.

2. *Semi-hairlessness.* Calves showing this mutation have a thin coat of short, fine, curly hair at birth, but later develop coarse, wiry hair which is never present in normal quantity. The coat appears patchy and the skin is wrinkled and scaly. Hair on the legs and back is longer and thicker than elsewhere. Semi-hairless calves seem to be wilder than normal ones.

This simple, recessive, autosomal mutation was studied in Polled Herefords in Oklahoma by Craft and Blizzard,[3] and later reappeared



RELATIONSHIPS OF THE "HAIRLESS" CALVES
Figure 10

It will be noted that the dam of one is also a daughter of the cow that produced the other. Both cows and the sire, III-3, are descended from the cow I-3.

in the same breed in California (Kidwell and Guilbert[8]).

3. *Hypotrichosis with anodontia.* Calves showing this mutation are completely hairless at birth, without even an eyelash. One of three studied by Drieux *et al.*,[3] acquired a downy coat of fine hair all over the body in its third week. Another developed hair between two and four months of age, the dorsal surface being covered earlier than the flanks and belly. All three were completely toothless at birth, and the one that was kept to six months of age had then only two first molars in the upper jaw. Because of this defect the calves had difficulty in handling anything but soft feed and did not thrive. Their tongues were longer and thicker than normal and protruded from their mouths. Sections of the skin showed malformation of the papillae at the bases of the hair follicles and cystic degeneration of the sweat glands, which lacked excretory tubules.

All three calves were males. The conclusion of the investigators that the causative gene was sex-linked and recessive is supported by the fact that the two cows which produced the three hairless calves were mother and daughter, while the sire of all three was unrelated, of a different breed, and the sire of over 180 normal calves by other cows.

4. *Viable hypotrichosis.* The mutation described in this paper differs from these others in being viable, with no effect on the teeth, and in causing a more complete degree of hairlessness than any except No. 3. Hair is confined to a few areas such as the inner surface of the ears, the eye-lids, and extremities of the tail and legs. The hair follicles lack papillae, and their cells are not differentiated into recognizable layers. The tubular (sweat) glands are cystic.

5. *Others.* In addition to these four different types of hypotrichosis, there is the defect of hair and incisor teeth found by Cole[1] in Holstein-Friesians. It was evidently hereditary, but the genetic basis was undetermined. In this case the coat of hair was less abnormal than in any of the four types listed above.

The partial hairlessness described by Liutikov[9] in crossbred cattle, partly Hereford, resembles the semi-hairless mutation in several respects and may have been the same. However, according to the description and the illustrations, Liutikov's calves had less hair over most of the body than those described by Craft and Blizzard, but in areas of the head, tail, legs and belly where the hair was white, the original coat did not fall out. Further information about this type of hypotrichosis is desirable.

Discussion

In some reports of hairless calves and reviews of the genetics of cattle, the writers have failed to recognize that all of these types of hypotrichosis are distinctly different from another hereditary abnormality known as epithelial defects, or *epitheliogenesis imperfecta.* In this condition, first studied by Hadley and Cole[6] in Holstein-Friesians, there are raw areas devoid of skin in some parts of the body, particularly on the lower half of the legs, on the inner surface of the ears and on the muzzle. In Ayrshires, Hutt and Frost[7] found the lesions to be much less extensive than in Holstein-Friesians, but in Jerseys the areas devoid of skin are so extensive[11, 15] that it is doubtful whether or not the condition could be caused by the same gene mutation as that in the other breeds.

In all three breeds, any hairlessness in the calves with epithelial defects is secondary to the loss of skin. Where the skin is normal, the hair is normal also. The distinction between this abnormality and the four kinds of hypotrichosis listed earlier is accentuated by the fact that some of the areas lacking skin in calves showing epithelial defects are the very ones most likely to carry normal hair in calves with lethal hairless, or with viable hypotrichosis. These are the extremities of the legs and the inner surface of the ears.

Summary

An extreme deficiency of hair was transmitted as a simple, autosomal, recessive character. The calf most severely affected lacked hair at eight weeks except on the end of the tail, inner surface of the ears, eyelids, lips, dewlap and umbilicus. At maturity the heifer had hair in these areas, also on the extremities of the legs, between the toes, and in lesser amounts elsewhere. Sections through hairless skin showed that the hair follicles lacked papillae and that their cells were not differentiated as in normal follicles. The tubular (sweat) glands were cystic.

The mutation is different from three other kinds of genetic hypotrichosis known in cattle: lethal hairless, semi-hairless, and hypotrichosis

with anodontia. The distinguishing characteristics of all four are briefly given.

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THE THIRD OUTER GLUME CHARACTER IN BARLEY*

Calvin F. Konzak*

A RECESSIVE gene which in the homozygous condition causes the formation of glume-like bracts on all central spikelets of the barley spike was reported in 1937 by Ivanova[1] in the U.S.S.R. This mutation characterized a black hulled, 6-rowed variety called *Hordeum vulgare var. afghanicum* by Vavilov. Ivanova established linkage of the third outer glume factor (now called *Trd trd*) with the factor *Bb* for black vs white color of the flowering glumes located on chromosome II. The percentage of crossing-over between the two factors varied from 15.35 to 16.94 in his studies.

Attempts to secure seed stocks possessing the "third outer glume" character for use in genetic studies in the United States have been without success.†

Recently, a plant possessing bracted spikelets was found by the author in a population of the variety Valki C.I. No. 5478. The white-seeded variety Valki originated at the Valkie Institute of Plant Industry in the U.S.S.R. but was pure-lined in the United States by G. A. Wiebe in 1941. Therefore, the new form could only have been the result of spontaneous mutation. Spikelets of the mutant form were bracted in a remarkably similar way to those of *H. vulgare var. afghanium*. The Valki mutant is shown in Figure 11, along with a black seeded segregate from the cross of the Valki mutant × Jet also possessing the mutant character.

Studies were undertaken to determine the relationship of the new bracted mutant to the "third outer glume" of Ivanova. Although it

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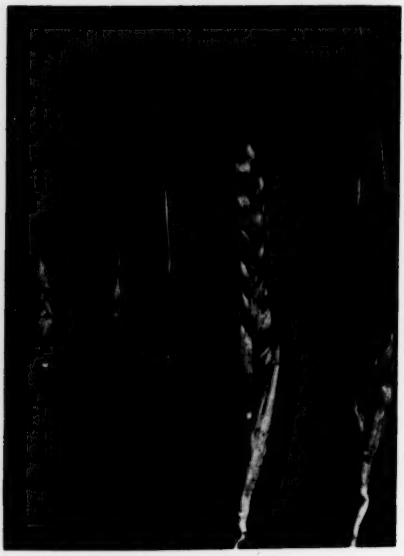
†Personal correspondence with Dr. G. A. Wiebe, Principal Agronomist in charge, Barley Investigations, U.S.D.A., Division Cereal Crops and Diseases, Beltsville, Maryland.



THE VALKI MUTANT
Figure 11

At the right are shown heads of the white-seeded bracted mutant, and a head of a black-seeded segregate from the cross of the Valki mutant × Jet. This also possesses the mutant character. At the left are shown central and lateral spikelets, showing details of the bracts.

was not possible to cross the bracted Valki mutant with Ivanova's *"trd,"* it was possible to collect comparable linkage data. Consequently, the black seeded variety Jet C.I. 967 was used as a parent in crosses with the bracted Valki mutant. Segregation of the characters bracted vs normal and black vs white glumes was recorded. A total of 157 lines were observed in F_3. All of the F_3 had been correctly classified in F_2, an indication that classification of the characters was not difficult. For this reason it was not considered necessary that the remaining F_2 population be observed in a succeeding generation.

The results obtained support the contention that the bracted Valki mutation is identical with the *"trd"* character of Ivanova (Table I). The difference in crossing-over percentage is entirely within the realm of experimental error. Linkage was calculated by the method of Immer.[2] Seed stocks possessing the *"trd"* character have been made available for use in genetic studies.

TABLE I. Summarized F_2 segregation in a cross of Jet × bracted Valki mutant

	Number				
	Observed	Calculated	Hypothesis	X^2	Probability Between
Non-bracted	425	411.75	3	.426	
Bracted	124	137.25	1	1.279	
Total	549	549		1.705	.20 .10
Black glumes	421	411.75	3	.203	
White glumes	128	137.25	1	.669	
Total	549	549		.872	.50 .30
Non-bracted, black glumes	384	308.81	9	18.307	
Bracted, black glumes	37	102.94	3	42.239	
Non-bracted, white glumes	41	102.94	3	37.270	
Bracted, white glumes	87	34.31	1	80.916	
Total	549	549		178.732	Less than .01*

*Indicates linkage; 14.2 percent crossing-over.

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(Received March 3, 1953)

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Atabrine as a De-Inhibitor of Radioactive Injury

. . . In a study designed to investigate modifications of the x-irradiation induced leucopenia in the chick, it has been observed that feathering is inhibited by irradiation and that this inhibition is overcome by adding Atabrine to the diet. . . .

At approximately 15 days after irradiation it was observed that the chicks that had received Atabrine were feathering at a faster rate than those not receiving Atabrine. The birds that had not received Atabrine produced apparently normal wing and tail feathers, but they developed very few feathers on the breast, back, and neck. . . .—Carl D. Douglass, Paul L. Day, Barbara Kelley and I. Meschan, *Science*, March 13, 1953.

ANURY IN COCKER SPANIELS

TERRY PULLIG*

IN two surveys of cocker spaniels during the summer of 1949 and 1950, the author was able to collect data on a number of physical characteristics one of which was a tailless condition. According to one breeder there was no sign of a tail in the ones that he saw, while another breeder stated that in one puppy there was only the first caudal vertebra. In the case that occurred in the author's kennel on June 5, 1944 there was no tail and other abnormalities were evident which will be discussed later.

Inheritance of Anury in Other Vertebrates

Congenital anury has been reported in many classes of the tailed vertebrates. In some cases taillessness was the result of a changed heredity, while in other cases occasional tailless individuals were produced by normal parents, but these tailless individuals evidently did not transmit this characteristic to their descendants.

Congenital taillessness in the rat which serves partially to bridge the gap between the so-called "hereditary" and the sporadic or accidental types was studied by Dunn *et al.*[4] They found tailless individuals in three different inbred families of rats. In one family there was a frequency of about 1.3 percent; in another family a frequency of about 1.6 percent and in a third family a frequency of about 3.7 percent. In each of these families taillessness occurred more frequently than in the general population of laboratory rats. In this material no single hereditary factor determined the difference between tailless and normal individuals. These workers stated that the genetic constitution determined the threshold of a reaction which was subject to alteration by minor and more or less random accidents early in development. Defects of a specific type were therefore more likely to occur in certain genetic constitutions than in others.

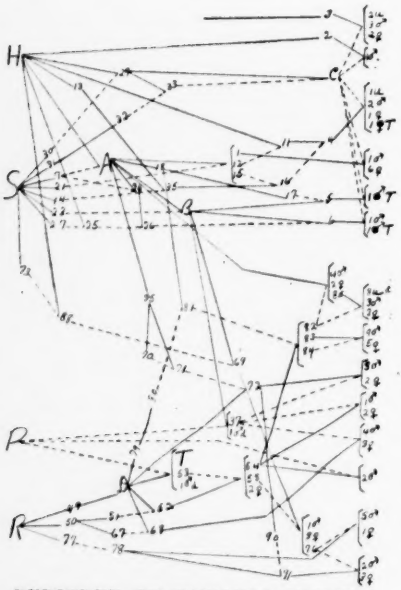
From the original short-tailed (brachy) stock following outcrosses to wild mice, three substrains have arisen, each characterized by complete absence of the bony tail. The tailless mice of each strain produced only tailless when bred to members of the same strain. In experiments with the pure breeding line of tailless mice (Line A), Chesley and Dunn[1] showed that such tailless mice contained two alleles, T and t^o, each of which was lethal when homozygous. The combination of these had the following effects: Tt^o produced tailless; TT produced the characteristically abnormal embryos which died about 11 days after fertilization; t^ot^o were abnormal embryos which died shortly after implantation; T- had short tails; and t^o- were normal.

Chesley and Dunn[1] found evidence that taillessness probably rested on a similar basis in the two other tailless lines (29 and 19), that is the brachyury lethal T might be balanced by a recessive lethal allele. The second allele might be different in the different tailless lines.

In breeding experiments with the true-breeding line of tailless mice (Line 29) Dunn and Gluecksohn-Schoenheimer[3] showed that the animals of this line are heterozygous for two lethal mutations; namely, T which was lethal to embryos at 11 days and t' which was lethal before implantation. Interaction between these mutations produced the tailless phenotype Tt'. Since no crossing over occurred between T and t', the genotype Tt' constituted a balanced lethal line and T and t' were assumed to be alleles.

Both the heritable and the non-heritable forms of taillessness are found in the domestic fowl. Dunn[2] suggested that rumplessness in fowls may be due to

*Aided by a grant from the Graduate School of Louisiana State University at Baton Rouge Louisiana.



PEDIGREE OF TAILLESS COCKER SPANIELS

Figure 12

The upper part of the pedigree shows an inbred family of cocker spaniels which produced heritable anury. The lower part is another family in which a "sporadic" tailless puppy occurred. (T = tailless, d = stillborn, and u = sex unknown.)

two distinct sets of causes, (1) a genic variation giving rise to the monofactorial dominant type of rumplessness, (2) an accident in embryogeny which arrests the development of the anlage of the caudal vertebra and tail at an early stage.

Description

In the upper part of Figure 12 the common ancestors of males 4, 5 and 6 and female C were males H and S. These dogs occurred once or twice "close up behind" both the sire and the dam of each of those four individuals who produced the three tailless offspring.

Most of the individuals in the lower part of Figure 12 are concerned with the matings made by the author, with the use of the author's stud by other breeders, and with the mating of bitches sold by the author. The first litter which was whelped in the research kennels at Louisiana State University had the only tailless individual that the author has ever seen (description elsewhere). This litter, which was by male D and out of female P, consisted of one normal male that was born dead, the tailless female T that died at one month of age, and the one normal female 53. The second mating of female P was to the male B (whose two sons in the upper part of Figure 12 produced anury when mated to female C) and this resulted in a normal stillborn male and the normal female 37. The last productive mating of female P was to her grandson, male 54, and produced two normal males. Female 53, the litter mate of the tailless puppy T, was bred to her half-brother, male 52, and produced all normal progeny. Lastly, she was purposely bred to a male that was not closely related to her and produced all normal offspring. Female 37, the half-sister of males 5 and 6 (males 5 and 6 produced anury when mated to female C) was mated to males 68 and 72 who are both half-brothers of the tailless puppy T and, lastly, to male 54 whose sire was also the half-brother of the tailless puppy T and the dam was the litter sister of the tailless female T. Female 55, whose sire was a half-brother of the tailless puppy T and whose dam was also the litter sister of the tailless female T, was mated to male 69 who is a half-brother of males 5 and 6 (males 5 and 6 produced anury when mated to female C) and all these offspring were normal. Female 82 was first mated to male A and then mated twice to her son out of her first litter and all these progeny were normal.

Despite all this intensive inbreeding of half-brothers to half-sisters, dam to son and litter brother to litter sister, there never occurred another tailless individual in the author's dogs. Based on these data, the author believes that there are two types of anury in cocker spaniels; one type which shows no evidence of a genic basis in contradistinction to the other type which shows ample evidence of a genetic background.

Anury in Cocker Spaniels

Concomitant with the taillessness condition in female T in the lower part of Figure 12, there was no anal opening; consequently, the exit for the feces was through the vulva. At one month of age the veterinarian made an internal exploration at which time he found that the large intestine was several times the size found in a normal puppy of this age. The large intestine was attached to and emptied by a small opening into the dorsoposterior end of the vagina. The tailless female failed to survive this drastic operation; thus, the author was left with only her dam, female P and her litter sister, female 53, with which to make a further study of the possible inheritance of anury in cocker spaniels.

On the other hand the taillessness in the three puppies in the upper part of Figure 12 was the only abnormality present. In the two tailless offspring by the half-brothers, males 5 and 6, according to the breeder, there was no indication of a tail and where the tails were supposed to arise, the surface was

perfectly smooth. The other breeder reported the tailless offspring by male 4 had one caudal vertebra. This tailless female was otherwise normal and was bred to her normal litter brother, but only whelped a single puppy which was normal.

Conclusions

Both types of anury are apparently relatively rare in cocker spaniels. Besides the four tailless puppies described in this paper, the author has heard of only two others having this condition.

The data, presented herein, seem to indicate that there is a non-heritable and a heritable type of anury in cocker spaniels. Apparently heritable anury in cocker spaniels is produced by a recessive mechanism.

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◆

PROBLEMS OF LIFE*

THIS book is an English Edition of a book published in Germany in 1949. A short review in a genetical journal seems appropriate as the author uses also the results of genetics to derive his point of view. This is what he has called the "organismic" point of view. He considers biology "an autonomous science in the sense that its problems require the development of specific conceptions and laws; further that biological knowledge and conceptions are active in different fields." This view is opposed to old fashioned mechanism which recognizes in the organism only the simple actions of physical laws as well as to vitalism which calls for a directing force. It is based upon the concept of the organism as a system in a steady state which requires definite physical conditions not found in the anorganic world.

In the first chapter the general aspects are discussed and the relation of the organismic concept to mechanism and vitalism worked out. The result is, that every organism represents a system i.e. a complex of elements in mutual interaction. From this it follows that it is impossible to resolve the phenomena of life completely into elementary units, for each individual part and event depends not only on conditions within itself, but also to a greater or lesser extent on the conditions within the whole or within superordinate units of which it is a part. Thus the whole can show properties that are absent from its isolated parts; the problem of life is that of organization. The leading principles of the organismic conception are therefore: "The conception of the system as a whole as opposed to the analytical and summative points of view; the dynamic conception as opposed to the static and machine-theoretical conceptions; the consideration of the organism as a primary activity as opposed to the conception of its primary reactivity."

These ideas (held by many biologists without putting them into a system) are tested with different groups of facts in the following chapters, dealing with physical and biological elementary units, cells and protoplasm and general principles of organization. Here the idea of "hierarchical order" is detailed, following mostly Woodger. It is here that the contact with genetics is made.

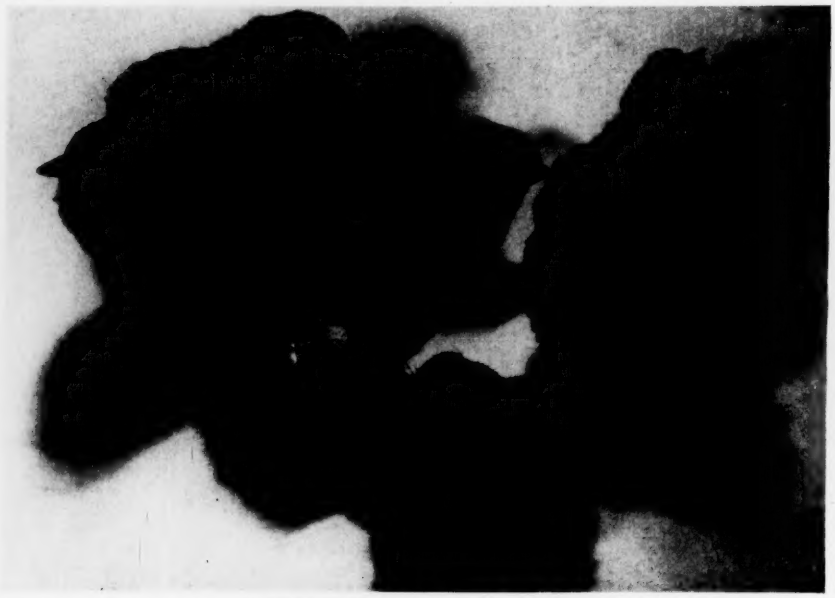
The next chapter on the unitary processes of life is the most important one for the geneticist. Here the question is analyzed how far genetically controlled development can be explained on physico-chemical principles. Using the reviewer's ideas it is shown that this is possible to a certain point after which some different principle is needed. It is discussed whether physical Gestalten would suffice i.e. systems that attain a state of equilibrium and represent physical wholes. This is again found insufficient in view of the facts of experimental embryology. At this point the author turns to the classical theory of the gene and comes to the conclusion that here too it is necessary to come from static to dynamic conceptions; inheritance is not a mechanism where genes are connected machine-fashion with the visible characters they produce, but rather it is a flow of processes in which the genes intervene in definite ways. Again this idea is developed

(*Continued on page 116*)

*L. von Bertalanffi. *Problems of Life.* An Evaluation of Modern Biological Thought. 1952. 216 pp. John Wiley and Sons, New York. $4.00.

"JITTERY," A SEX-LINKED NERVOUS DISORDER IN THE CHICK

ELWARD F. GODFREY, B. B. BOHREN AND R. GEORGE JAAP*



A GROUP OF "JITTERY" CHICKS

Figure 13

Seven "Jittery" chicks, showing the various degrees of head retraction associated with the condition. This picture does not show the typical rapid sidewise vibration of the head.

FOUR inherited nervous disorders of the fowl have been reported in the literature. The phenotypic manifestations of the condition described herein, which is apparently the fifth congenital nervous disorder of the fowl to be reported, partially resembles two other inherited nervous disorders, but differs from both in mode of inheritance.

Knowlton[4] described a condition in the newly hatched chick which he designated as "congenital loco" (*lo*). The condition behaved as an autosomal recessive. Affected chicks usually could not stand more than a few seconds; the head was drawn back and the beak was directed upwards. Knowlton (*loc. cit.*) attempted to raise some of the "loco" chicks but all died within nine days post-hatching, probably because of their inability to eat and drink. Hutt and Child[3] reported the presence of a semi-lethal hereditary condition in young chicks which was characterized by tremors.

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The intensity of the tremors declined after the second day, but most of the afflicted chicks died. The condition, which was designated "congenital tremor," appeared to behave as a simple autosomal recessive with low penetrance. The third inherited nervous disorder of the fowl has been described by Scott, *et al*.[7] "Shaker" chicks were normal at hatching, but developed a characteristic mild, rapid movement of the head and neck by four weeks of age. The symptoms became more severe with advancing age and most of the "shaker" chicks died from inanition, although a few were reared to sexual maturity. The condition was attributed to a degenerative loss of Purkinjie cells of the cerebellum. Goodwin, *et al*.[2] described another condition which is not present in the day-old chick, but which was almost completely lethal during the early growing period. Affected chicks suddenly became comatose, and occasionally showed dyspnea or tetanic contractions, which resulted in death. However, a few chicks were able to survive after one or two attacks. The condition was shown to be due to a sex-linked recessive gene (*xl*). The "jittery" condition, described herein, apparently adds another to the group of genes known to cause nervous disorders in the fowl. A preliminary report of this condition has been presented by Bohren.[1]

Phenotypic Expression

Afflicted chicks are characterized by a tendency for the head to be retracted over the back, accompanied by a rapid sidewise vibration of the head. The intensity of both symptoms is somewhat variable. The chicks have difficulty in standing; they often fall over backward and will remain on their sides until righted, after which the entire performance is repeated. Startling the chicks tends to increase the intensity of both of the "jittery" symptoms.

The condition is almost completely lethal. Tennessee data indicated that approximately 75 percent of affected chicks die during the first two days post-hatching, while nearly 90 percent are dead at the end of the first week. Only one to two percent survive to sexual maturity. No affected males have been reared to functional maturity. Death appears to result from starvation, and those that survive exhibit various degrees of emaciation. Mature survivors tend to move in circles, while exhibiting a notable lack of coordination of the head and neck. As with affected day-old chicks, fear or excitement tends to increase the intensity of the "jittery" characteristics in mature birds.

Genetics

The "jittery" condition was observed at Purdue and Ohio State Universities in the progenies of a Single Comb White Leghorn and a New Hampshire male respectively. In each case the suspicious males were mated to normal females in order to investigate the mode of inheritance of the disorder. Further studies of the Ohio mutant have been carried on at the Tennessee Experiment Station. The combined results at the three stations are shown in Table I. With the exception of the seven males classified as jittery, the data seem to fit the hypothesis that this condition is due to a sex-linked gene. All such affected males were classified at the Ohio and Tennessee stations. It was observed that these male chicks were progeny of the original Ohio New Hampshire carrier male and some of his Tennessee sons and grandsons. It is possible that the original male as well as some of his progeny were heterozygous for the congenital loco gene as well as jittery. When, by chance, they were mated to apparently normal females which were also heterozygous for the loco gene, some males were produced showing the loco phenotype, which would be readily misclassified as jittery.

It was also observed that in general the symptoms exhibited by the jittery progeny of the Ohio male were more severe than those observed at Purdue University. This greater penetrance might have resulted in occasional heterozygous male chicks exhibiting jittery symptoms. At the Tennessee Station, however, the severity of the condition as observed at Ohio decreased until it approached the intensity observed at Purdue. Some affected male chicks were observed at Tennessee in spite of this fact.

Other possible explanations for these exceptional chicks could be that differential embryonic mortality occurred or that a partial

TABLE I. Classification of chicks obtained from mating of normal male (heterozygous jittery) × normal females

	Jittery		Normal	
Sex	♂	♀	♂	♀
observed	7	155	341	172
expected	0	168.75	337.50	168.75

TABLE II. Distribution of progeny from mating heterozygous normal ♂ (Purdue) × jittery ♀ (Purdue)

	Jittery		Normal	
Sex	♂	♀	♂	♀
observed	6	2	5	4
expected	4.25	4.25	4.25	4.25

sex-limited type of inheritance was involved. The hatchability obtained from these matings (68 percent of fertile eggs set) and the equality of the sex-ratio would tend to invalidate the first possibility. In order to test the possibility of a sex-limited expression of this gene, a normal but carrier male was mated to jittery females at Purdue. The results are shown in Table II. The small numbers are due to the few affected females available and their low fecundity. In spite of the small number of offspring, it is evident that homozygous males of the Purdue line do exhibit the condition in the expected ratio ($\chi^2 = 2.06$, P = >. 50). Thus, while no definite conclusion may be drawn, the evidence points to the presence of the congenital loco gene at a low frequency in the Ohio-Tennessee line.

If the seven affected males shown in Table I are considered to carry the loco gene and thus are included with the normal males, the ratio closely approaches that expected for a sex-linked recessive gene ($\chi^2 = 1.45$, P = .30-.50). If the gene were not sex-linked, equal numbers would be expected in each of the classes which would deviate significantly from the observed data ($\chi^2 = 346.6$). Further evidence for sex-linkage is the fact that carrier males produced jittery female offspring regardless of the source of the female parents, and that all individual females to which such males were mated produced jittery female offspring. Thus the data indicate quite clearly that jittery is inherited as a sex-linked recessive gene.

The Relationship of the Ohio and Purdue Mutants

When originally observed, the Ohio mutant was more severe in the expression of jittery symptoms than was the Purdue mutant, particularly with respect to head retraction. A normal male heterozygous for the Ohio gene was mated to jittery females carrying the Purdue mutant. The distribution of the progeny as shown in Table III indicates that the genes involved at the two stations are the same ($\chi^2 = 2.6$, P = .30-.50). The phenotypic differences in the severity of expression therefore must be due to modifying factors. This is further substantiated by the fact that as the Ohio stock was carried through succeeding generations at the Tennessee Station, the expression became less severe.

Linkage Relationships

It is not difficult to produce males heterozygous for jittery, so three-point linkage tests were conducted at the three stations. For the other two loci, barring and rapid-feathering were chosen since both are readily identified in the day-old chick and are nearly at opposite ends of the known sex-linked group. With only a few jittery females available, it was necessary to mate the triple heterozygous males to normal females. In this case, only the female offspring could be classified for linkage. Furthermore, the genotypes of the female parents would not be important since they would affect only the male progeny. Black Australorp females were used at Purdue, Black Minorcas at Ohio State, and White Plymouth Rocks and New Hampshires at Tennessee. All chicks were classified at hatching and sex was determined by dissection.

At Purdue, the jittery gene occurred in a White Leghorn male which was mated to New Hampshire females. The jittery chicks from this mating were females carrying rapid-feathering, jittery, and some or all carried barring. These females were then mated to Black Australorp males which were homozygous for slow feathering, non-jittery, and non-barred. The barred offspring of this cross were males heterozygous for the three genes in question with their genotypes being *Bkj/bKJ*. The Ohio and

TABLE III. Results of mating a normal ♂ heterozygous for the Ohio mutant × Purdue jittery ♀

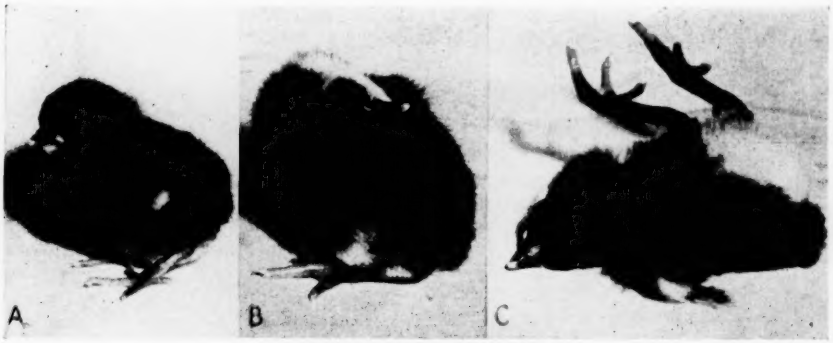
	Jittery		Normal	
Sex	♂	♀	♂	♀
observed	7	9	14	10
expected	10	10	10	10

TABLE IV. A—Classification of female chicks from Purdue mating of slow, barred, normal males (*Bkj/bKJ*) × slow, non-barred, normal females (*bKJ/—*)

Gametes	No. Chicks	Total	Expected if random
rapid, bar., jit.	54		
slow, non-bar., normal	73	127	126
rapid, bar., normal	73		
slow, non-bar., jit.	65	138	126
rapid, non-bar., normal	55		
slow, bar., jit.	56	111	126
rapid, non-bar., jit.	48		
slow, bar., normal	80	128	126

B—Classification of female chicks from Ohio-Tennessee matings of slow, barred, normal males (*BKJ/bkj*) × rapid, non-barred, normal females (*bkJ/—*).

Gametes	No. Chicks	Total	Expected if random
rapid, non-bar., jit.	37		
slow, bar., normal	48	85	75.25
rapid, non-bar., normal	38		
slow, bar., jit.	36	74	75.25
rapid, bar., normal	41		
slow, non-bar., jit.	28	69	75.25
rapid, bar., jit.	31		
slow, non-bar., normal	42	73	75.25



NORMAL AND "JITTERY"

Figure 14

A—Shows a normal chick. *B*—A typical "Jittery" chick, showing the most extreme form of head retraction. *C*—The typical position of a "Jittery" chick after it has fallen over.

Tennessee triple heterozygous males were somewhat different with their genotypes being *BKJ/bkj*.

The results of these tests are shown in Table IV A and B. In neither case do the data deviate significantly from random distribution ($\chi^2 = 2.97$, P = .30-.50, and $\chi^2 = 1.87$, P = > .50). The first group in each table is theoretically the non-crossover gametes. This group is the largest, as expected, in Table IV B but not in Table IV A. It is interesting, however, that the third pair of gametes in each case is the smallest group. In both tables it is the *k* gene which has changed position in these groups as compared to the non-crossover gametes. This result might be taken as meager evidence that these groups are the double crossover gametes and that the *k* gene is located in the middle. This result is of further interest in view of the data of Mueller[5] which indicate that the gene for "shaker" is located approximately 26 units from the *k* locus and about 35 units from the silver (*S*) locus on the sex chromosome. Linkage tests of the jittery gene with that for shaker, as well as other sex-linked genes which have not been mapped (naked, albino, dwarfism, *xl*), would be enlightening.

A summary of the data by pairs of genes is shown in Table V. The 47.3 percent crossing over observed between the *B* and *K* loci was only slightly greater than the 46 percent reported by Punnett.[6] None of the three comparisons differs significantly from a 1:1 ratio ($\chi^2 = 0.696$, P = .30-.50). These linkage results are open to suspicion, however, since a highly significant deficiency of jittery chicks and a near significant deficiency of rapid feathering chicks occurred as shown by a single gene classification (Table VI). The reason for these deficiencies is not apparent since no such deficiencies were observed in previous matings (see Table I) and the sex ratio among the progeny of these matings was normal (818 males : 805 females).

It is unlikely that the jittery gene could lie between *B* and *K* and still show

TABLE V. Two-gene classifications of linkage test results. Data from all stations combined

Genes Tested	Total Gametes	Parental Types	Crossover Types	Crossover Percent
B and *K*	805	424	381	47.3
K and *J*	805	413	392	48.7
B and *J*	805	392	413	51.3

TABLE VI. Single gene classification of linkage test data (combined)

	Rapid	Slow	χ^2	P
observed	377	428		
expected	402.5	402.5	3.10	.05-.10
	Barred	Non-barred		
observed	419	386		
expected	402.5	402.5	1.27	.20-.30
	Jittery	Normal		
observed	355	450		
expected	402.5	402.5	10.98	<.01

linkage with neither. If located distally on the chromosome from either of the other genes it is some distance away (Table V) and the sex chromosome linkage map must be longer than has been indicated by previous evidence. The slight evidence of double crossing over with rapid feathering as the middle gene would suggest that the order for these three genes may be *B-K-J*.

Physiological Basis

Histological examination of "jittery" chicks indicated that most exhibited a marked deterioration of the Purkinjie cells of the cerebellum. These cells appeared to be fewer in number than normal, and 10 to 50 percent of the existing cells stained more intensely than normal. There appeared to be little relationship between the severity of the physical symptoms and the amount of deterioration of the Purkinjie cells although no changes in number or staining intensity of the Purkinjie cells were noted in the normal controls which were examined. Scott, *et. al*[7] have reported that "shaker" chicks examined histologically exhibited a significant loss of Purkinjie cells accompanied by a degradation of those cells remaining. In view of these findings, it would appear that the physiological basis of the "jittery" and "shaker" conditions are similar.

Summary

A condition in day-old chicks referred to as "jittery", in which the head is retracted over the back, accompanied by a rapid shaking of the head has been studied. The character is highly lethal in post-embryonic stages with approximately one to two percent of affected females reaching maturity. In adult hens the expression of the gene is similar to that in chicks and in addition they tend to circle rapidly when frightened. Breeding tests show the gene for jittery to be a sex-linked recessive. Males heterozygous for jittery, barring and rapid feathering were mated to normal females to test for linkage. Since the non-jittery gene of the females is dominant, only the female offspring were studied. A total of 805 female progeny were classified. The only evidence of linkage was a slightly reduced number of one type of gametes which may be the double-crossover class. If so, this would suggest that the rapid feathering gene is in the middle and that the probable order of these genes is *B-K-J*. It appears that the gene for jittery is located distally on the chromosome from the other genes involved; this suggests that the sex-chromosome map of the fowl may be longer than presently described.

Histological examination of affected chicks revealed marked degeneration of the Purkinjie cells of the cerebellar region.

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HEREDITARY UMBILICAL HERNIA IN HOLSTEIN CATTLE

J. P. W. Gilman and E. W. Stringam*

HEREDITARY hernias have been reported as occurring in the inguinal, scrotal and umbilical regions of most breeds of domestic animals. In a recent extensive review of this subject, Kobozieff and Kobozieff[3] cite cases of umbilical hernia of a hereditable nature in horses, cattle, swine, dogs, cats and goats. This condition, although comparatively rare, has also been reported by Moore and Schaible[5] as occurring as a simple recessive characteristic in the laboratory rat.

Studies concerned with umbilical hernia of cattle have given rise to several conflicting hypotheses as to the nature of the hereditary factors involved. Warren and Atkeson,[8] reporting the occurrence of this defect in the Holstein breed in the U.S.A., suggested that the factor involved was either a sex-limited dominant, by which females were not affected, or possibly sex-influenced, being dominant in the male and recessive in the female. Wriedt[9] has stated that the condition is transmitted most frequently as a simple dominant although he also mentions that some cases of umbilical hernia appear to result from the action of recessive genes.

Methods

The present study was undertaken when it was noticed that the number of herniated calves presented at the Ontario Veterinary College clinic had been increasing progressively since 1946, and that the vast majority of all such cases involved purebred Holstein cattle. On further examining these records, it was found that a great many of the calves were of a similar line of breeding, tracing back within two or three generations to a certain bull "M" or his sire "P".[1]

A survey was undertaken in co-operation with the Holstein breeder-members of a nearby artificial insemination unit, to ascertain, amongst other things, the incidence of umbilical hernia in the progeny of their seven Holstein bulls. A record of calves born of artificial insemination during a seventeen month period was obtained from this source, and wherever possible these animals were examined physically by the investigators. However, such examinations were almost entirely restricted to the female half of the population, as the young bull calves had in most instances been sold for vealing at a very early age.

The data analyzed in the ensuing report were restricted to those obtained from herds for which complete records were available. Any umbilical sacculation, regardless of size, that could be demonstrated on palpation to communicate with the abdominal cavity was recorded as a hernia.

Male No. 1 of the seven "Unit" bulls had been operated on for a congenital umbilical hernia prior to his purchase by the Unit. Bulls No. 2 and 3 had as their common sire bull "M" which was also the paternal grandsire of bull No. 1. Furthermore, this last mentioned bull's maternal grandsire was bull "P." Two out of the remaining four bulls (6 and 7) did not trace back directly to either bulls "P" or "M" whilst the fourth animal had bull "P" as his paternal great-grandsire and the fifth animal carried only about one sixteenth of the genes of this ancestor (Table 1).

Results

Table I summarizes the information obtained in this study from those herds for which com-

*A contribution from the Section of Embryology and Genetics, Ontario Veterinary College, Guelph, Canada, and the Animal Husbandry Department, Ontario Agricultural College, Guelph, Canada. The authors are grateful for the wholehearted co-operation received from both the officers and members of the Waterloo Cattle Breeding Association, Waterloo, Canada, without which this study would not have been possible.

plete breeding records were available. Two thousand and forty-five calves were listed, of these 1,047 were heifers, while 998 were bulls, which does not constitute a significant deviation from the expected 50:50 sex ratio.

Sixteen cases of congenital hernia occurred among these calves, giving an incidence of 0.79 percent. This frequency was not found to be evenly divided between the sexes, there being a 1.06 percent incidence in females against only 0.51 percent in males. It should be emphasized that very few of the male calves were actually examined by the investigators, for reasons given previously, and that a small umbilical hernia might easily be overlooked by the owner in the very young calf. For this reason the data pertaining to bull calves were considered unreliable and the ensuing analysis has been confined to the female members of the herds.

The eleven herniated heifers were found to be a portion of 628 female calves sired by bulls 1, 2 and 3. The remaining bulls sired a total of 419 heifers none of which was observed to have umbilical hernia (Table I). This difference is highly significant ($P<0.01$).

Assuming bull No. 1 to be homozygous for the hernia factor, either a single or two factor recessive gene may be postulated as being responsible for the characteristic. In the former case, bull Nos. 2 and 3 would be expected to leave half as many herniated offspring as would bull No. 1, among equal numbers of progeny out of a similar female population. No significant deviation from this expectation existed ($\chi^2 = 0.7363$; df $= 2$; $P > 0.7$). In the latter case, if two pairs of recessive factors were involved, the carrier bulls would be expected to leave either ¼ or ½ as many hernias as would the homozygous double recessive bull No. 1, depending upon whether both carriers were completely heterozygous or whether one or the other of them was homozygous for one pair of factors and heterozygous for the other. Table II demonstrates that there was no significant deviation from any of these three alternatives.

Discussion

Roberts and Yapp[6] and Roberts[7] report a sex ratio of 49.4 percent males among all births in a sample of 13,000 cattle under natural breeding conditions. These authors reported considerable variation between different breeds, however the sex ratio of 48.6 percent males reported by them for the Holstein breed in the U. S. A. is in very close accord with the ratio of 48.8 percent males reported here for artificially inseminated Holsteins. The question has been raised as to whether or not the A.I. technique may tend to bias the sex ratio in favor of males and a report by Kafka[2], comparing ratios between bulls used both naturally and artificially, gives a proportion of 48.39 percent males under the former and 53.4 percent males under the latter conditions. This author suggests that specific bull differences are more probably responsible for the excess of males than is artificial insemination as a method. However his conclusions are based on very small numbers. An examination of the sex ratios from the individual bulls listed in Table I shows that biased ratios tend to appear for those animals for which only small numbers of progeny are recorded. It seems almost certain that such observed differences as these are not real, but are examples of that "chance deviation" which becomes increasingly common when numbers are small, as has frequently been emphasized by Lush[4] and other authors.

Despite the fact that twice as many heifers as bulls were recorded as being born with hernia, there was no evidence

TABLE I. Summary of the breeding records of seven Holstein bulls with their relationship to a common ancestor

Sire No.	Relationship to Bull "P" Direct	Total*	Progeny & Percent Hernia Males	% Hernia	Females	% Hernia
1	37.5	46.9	214	1.4	225	3.3
2	37.5	45.3	238	0.42	251	1.2
3	25.0	37.5	123	0.81	152	0.7
4	12.5	14.1	104	0	120	0
5	6.2	29.3	69	0	55	0
6	0	10.9	88	0	75	0
7	0	6.2	162	0	169	0

*Calculated after the method described by Wright[10] using five generation pedigrees.

TABLE II. Probabilities for various two-factor combinations in the transmission of umbilical hernia by three carrier bulls

Ratio	No.	Genotype	Phenotype	Obs.	Exp.*	χ^2	P
A							
	1	uu u′u′	Hernia	7	7.60		
4:1:1	2	Uu U′u′	Normal	3	2.12		
	3	Uu U′u′	Normal	1	1.28	.4740	0.70
B							
	1	uu u′u′	Hernia	7	6.80		
4:1:2	2	Uu U′u′	Normal	3	1.90		
	3	uu U′u′	Normal	1	2.30	1.3775	0.5
C							
	1	uu u′u′	Hernia	7	6.37		
4:2:1	2	Uu u′u′	Normal	3	3.55		
	3	Uu U′u′	Normal	1	1.08	.1534	0.80

*Assuming a heterozygous female population, with calculations based on total hernias occurring in the cow population bred to the three bulls and weighted for the proportion of cows to each.

of any association between this factor and an embryonic sex-linked lethal affecting a proportion of the males. That this unequal sex distribution of the defect was not just a chance result of the comparatively small number of affected animals observed, is supported by the fact that among a sample of 91 herniated calves, operated on at the Ontario Veterinary College clinic during the past few years, the sex ratio was exactly 2.5:1 in favor of heifers. The possibility of this being the result of the additive effect of modifying genes carried on the sex chromosome and influencing umbilical hernia should not be discounted, particularly as the extreme variability in expression observable between umbilical hernias suggests that numerous such factors may be involved in their development. However, here again in the absence of definite evidence to the contrary, it would seem that the most likely explanation of the disparity between the sexes is the small number of bull calves saved for breeding purposes. If this is so, then the incidence of hernia as reported here is at least 25 percent lower than that which actually existed in the sample studied.

Either a sex-limited or sex-influenced mode of inheritance for the factor responsible for umbilical hernia, as suggested by Warren and Atkeson[7], is directly opposed to the ratios observed in this investigation. One can only assume that either these investigators were dealing with genetic factors for hernia other than those under study here or that their ratio of 20 male : 1 female was a chance deviation from an actual 50-50 distribution. Their data were confined to the progeny of one bull used on two Holstein herds.

The indications are, from the present study, that either one or more pairs of autosomal recessive factors, of rather low frequency, are responsible for umbilical hernia. That this frequency might easily increase to economically important proportions in a relatively short period of time, through the inadvertent aid of artificial insemination, is clearly indicated from the conditions revealed in the present study. Reference to Table II shows that a very good fit exists for the two factor hypothesis of a 4:2:1 ratio, assuming bull No. 1 to be completely homozygous, bull No. 2 to be homozygous for one pair and heterozygous for the other and bull No. 3 to be completely heterozygous. This observation is the more interesting because bull No. 1 was known to have had a hernia at birth, while bull No. 2 has 50 percent higher concentration of germ plasm from the common carrier ancestor male "P" than has bull No. 3.

Summary

The progeny of seven Holstein bulls in service at an artificial insemination unit were checked for umbilical hernia and sex ratio. Data for 2,045 calves showed a sex ratio of 48.6 percent males with individual bull differences being largest in sires with comparatively few progeny.

The data concerning hernia were restricted to the 1,047 females of the sample as a majority of the males were not actually examined. This latter fact was considered the most probable explanation for the recorded incidence of hernia among heifers being over twice as high (1.06 percent) as that for bulls (0.51 percent).

Hernias were restricted to the progeny of three of the seven bulls, those showing 25 percent to 37.5 percent direct relationship to a common ancestor previously suspected (from clinical observations) of being a heterozygous carrier. The four bulls that did not leave any herniated offspring ranged from 0 to 12.5 percent relationship to this ancestor.

It was concluded that either one or more pairs of autosomal recessive factors of rather low frequency were responsible for the occurrence of this undesirable characteristic.

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(*Received January 26, 1953*)

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Problems of Life

(*Continued from page* 107)

with the reviewer's theories of harmonized reaction-velocities in development. This leads to the theory of the gene which the author wants to be looked at in an organismic way. Actually this is found in the reviewer's ideas on the nature of the gene as developed since 1937. The author could have added here that this modern development of the theory of the gene is a very good illustration of the hierarchical principle of organization (see above Woodger) which is bound to lead to further insight.

This leads to a discussion of evolution. Many aspects are reviewed with the conclusion that evolution is not a process at random, but is governed by definite laws, still to be discovered. The reviewer considers this chapter less well organized than the rest of the analysis. It finishes with a discussion of what Boveri once called: the organism as a historic being. Here the statement is found: "In a unitary conception of the basis of heredity, it may be possible to interpret phylogenetic changes not as an adding of new genes but rather as a transition to a new state in the genome as a whole: in a similar way that psychological memory is to be interpreted rather as a change in the whole 'brain field' than as the accumulation of separate and individual traces." This is, of course, essentially the type of evolutionary thinking which the reviewer has proposed since 1933.

The last chapters on the laws of life, life and knowledge and the unity of science contain the general philosophical elaboration, especially comparing organisms and non-organized matters. They can hardly be reviewed in a short space but are eminently worth reading for anybody who tries to put order into his ideas concerning living and non-living matter. Here also the related ideas of Schrödinger and Pascual Jordan are discussed.

This review in a genetical journal seems to be justified by the fact that more and more of the younger generation of scientists specialize so early and so completely that they are not able any more to conceive or even to understand ideas which are based upon a broad knowledge of the entire field. The result is frequently an unsound skepticism towards all constructive thinking and a fanatical partisanship for whatever is the fashionable theory in the narrow field known to them. It would be a good thing for this type of specialist to stop occasionally and to read a thought provoking book as the one reviewed, which looks at biology from above, not from below.

RICHARD B. GOLDSCHMIDT

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Berkeley

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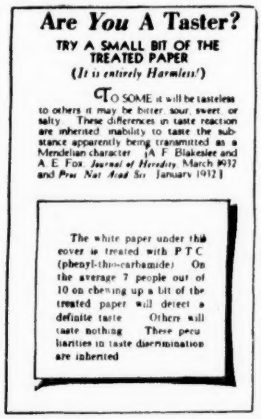
Corrigendum

TO THE EDITOR:

With reference to my paper A GENE FOR YELLOWISH-WHITE DOWN IN THE RED JUNGLEFOWL which appeared in the *Journal of Heredity*, XLIV (2) 1953, I wonder if a very important typographical error could be noted in the May-June issue of the Journal. On page 51—toward the end of the second paragraph—after referring to Dunn, Grüneberg, and Snell on genetic symbolization, the gene symbol should read lower case "*st+*" not capital "*St+*." For example, ". . . then the symbol *c+* or *st+* (for *wild type* striping) should stand as the etc. . . ."

G. VICTOR MOREJOHN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